Hemodynamics of Shock and MCS

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Disclosures

- None pertaining to this talk

Outline

- Definition
- Epidemiology
- Pathophysiology
- Hemodynamics
- Management
- Mechanical support devices

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Table 2: Different Definitions of Cardiogenic Shock

Clinical Definitions	European Society of Cardiology[89]	SHOCK Trial[6]	IABP-SHOCK II[87]	CULPRIT SHOCK[36]
Ineffective cardiac output due to primary cardiac dysfunction resulting in inadequate end-organ perfusion Cardiac disorder that results in both clinical and biochemical evidence of tissue hypoperfusion A clinical condition of inadequate tissue (end-organ) perfusion due to cardiac dysfunction	Clinical criteria: SBP <90 mmHg with adequate volume and clinical or laboratory signs of hypoperfusion Clinical hypoperfusion: cold extremities, oliguria, mental confusion, dizziness, narrow pulse pressure Laboratory hypoperfusion: metabolic acidosis, elevated serum lactate, elevated serum creatinine	Clinical criteria: acute MI complicated by left ventricular dysfunction SBP <90 mmHg for >30 min or support to maintain SBP >90 mmHg and end-organ hypoperfusion (urine output <30 ml/h or cool extremities) Hemodynamic criteria: cardiac index <2.2 l/min/m^2 and PCWP >15 mmHg	Clinical criteria: acute MI SBP <90 mmHg or >30 min or catecholamines to maintain SBP >90 mmHg and clinical pulmonary congestion and impaired end-organ perfusion (altered mental status, cold/clammy skin and extremities, urine output <30 ml/hour, or lactate >2.0 mmol/l)	Clinical criteria: SBP<90mmHg for longer than 30 min or Catecholamine therapy to maintain a SBP >90 mmHg, clinical signs of pulmonary congestion, and signs of impaired organ perfusion with at least one of the following manifestations: altered mental status; cold and clammy skin and limbs; oliguria with urine output <30ml/h; or arterial lactate level >2.0 mmol/l

PCWP = pulmonary capillary wedge pressure; SBP = systolic blood pressure.

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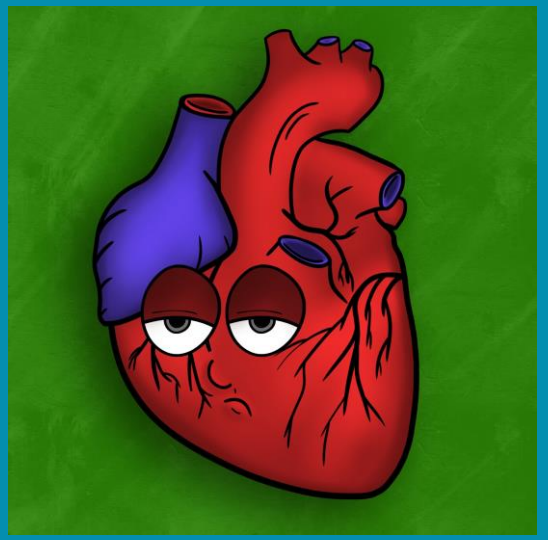
Cardiogenic Shock Management and Research: Past, Present, and Future Outlook, *US Cardiology Review 2022;16:e03.*

Definition

- LOW CARDIAC OUTPUT
- TISSUE HYPOPERFUSION

Severe impairment of myocardial performance leading to diminished cardiac output, end organ damage, and hypoxia.

CS carries marked morbidity and mortality, with limited data to guide hemodynamic targets.



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Epidemiology

- AMI- CS-Acute myocardial infarction (MI) accounts for > 80% of patient in CS.
- CS complicates 5% to 10% of cases of acute MI
- Leading cause of death after MI.
- STEMI – 2X risk

- NSTEMI-associated CS are less likely to undergo early cath, delaying PCI and/CABG and increasing the risk of mortality compared with patients with STEMI-associated CS.
- Chronic HF related CS
- Higher CS in
  - women
  - Asian/Pacific Islanders
  - aged >75 years.

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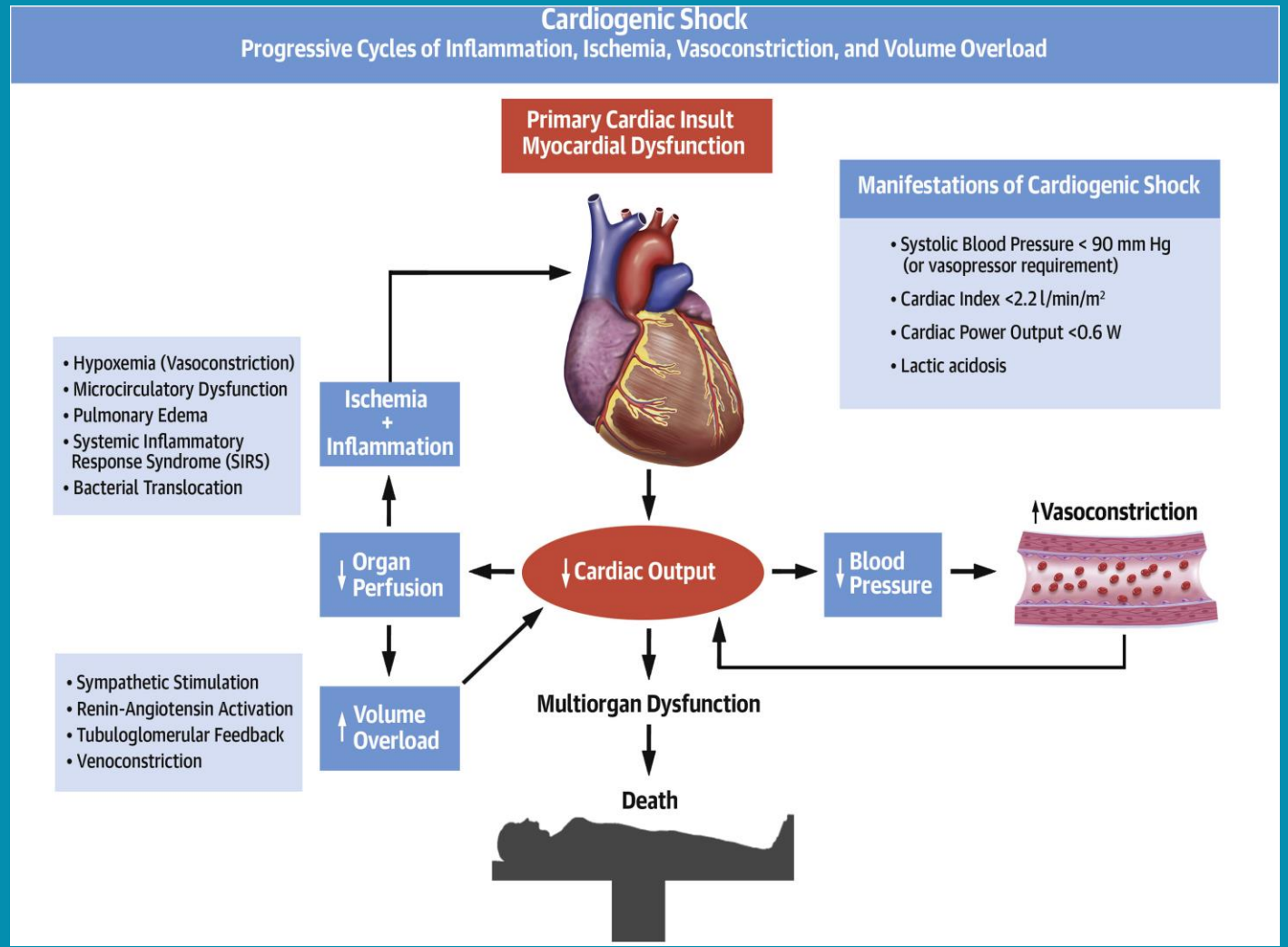
Pathophysiology



Tehrani B, Truesdell A, Psotka M, et al. A Standardized and Comprehensive Approach to the Management of Cardiogenic Shock. *J Am Coll Cardiol HF.* 2020 Nov, 8 (11) 879–891.

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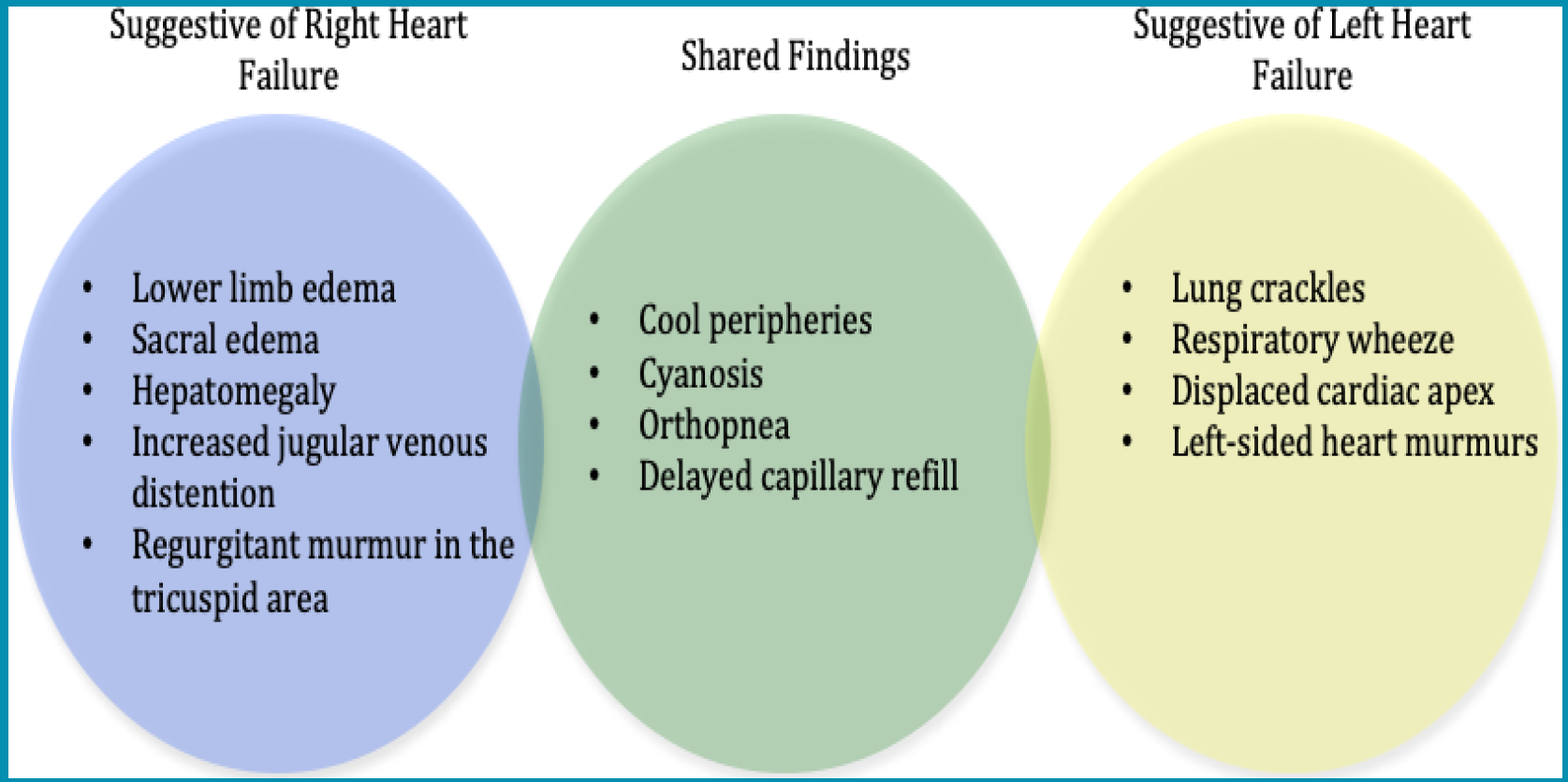
Clinical Presentation and Physical Examination



Cyrus Vahdatpour. Journal of the American Heart Association. Cardiogenic Shock, Volume: 8, Issue: 8, DOI: (10.1161/JAHA.119.011991)

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Clinical Presentation and Physical Examination

		Volume Status	
		Dry	Wet
Peripheral Perfusion	Warm	**Vasodilatory shock** (not CS) Increased cardiac index, low SVRI, low/ normal PCWP	**Mixed CS** Low cardiac index, low / normal SVRI, Elevated PCWP
	Cold	**Euvolemic CS** Low Cardiac index, high SVRI, low / normal PCWP	**Classic CS** Low cardiac index, High SVRI, Elevated PCWP

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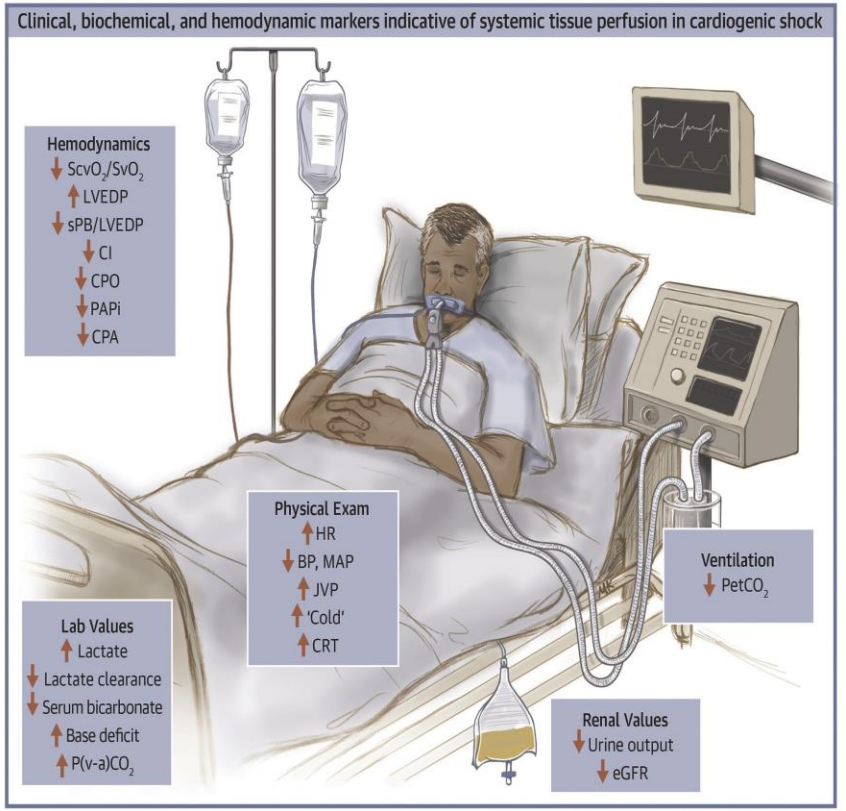
Initial Investigation

- EKG
- CBC/BMP
- CARDIAC ENZYMES
- NT-BNP
- PO2/PCO2
- LACTIC ACID/BICARB
- ECHO
- CATH/REVASCULARIZATION

CENTRAL ILLUSTRATION: Clinical, Biochemical, and Hemodynamic Variables



Mathew R, et al. JACC Adv. 2022;1(2):100034.

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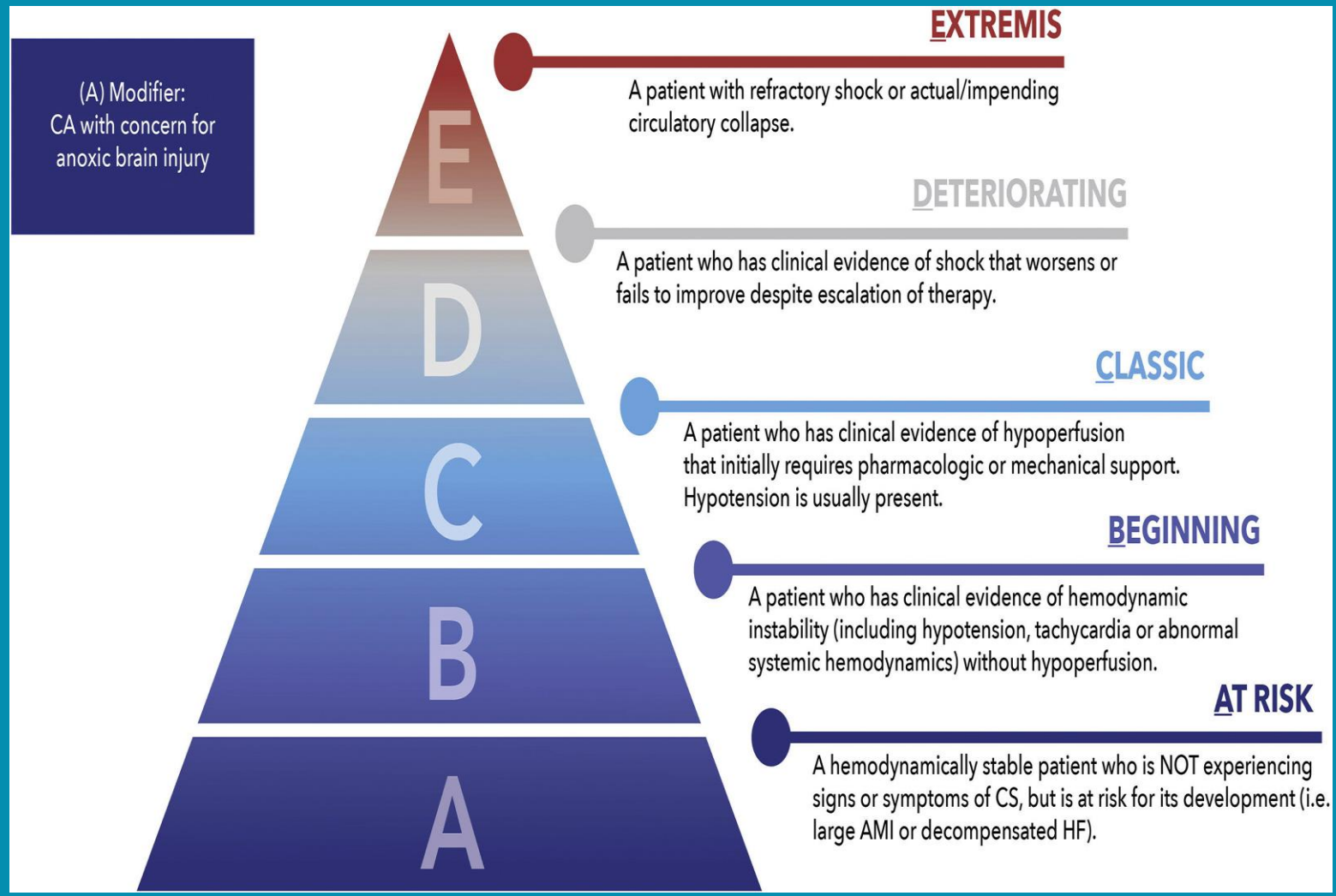
SCAI SHOCK Classification Pyramid



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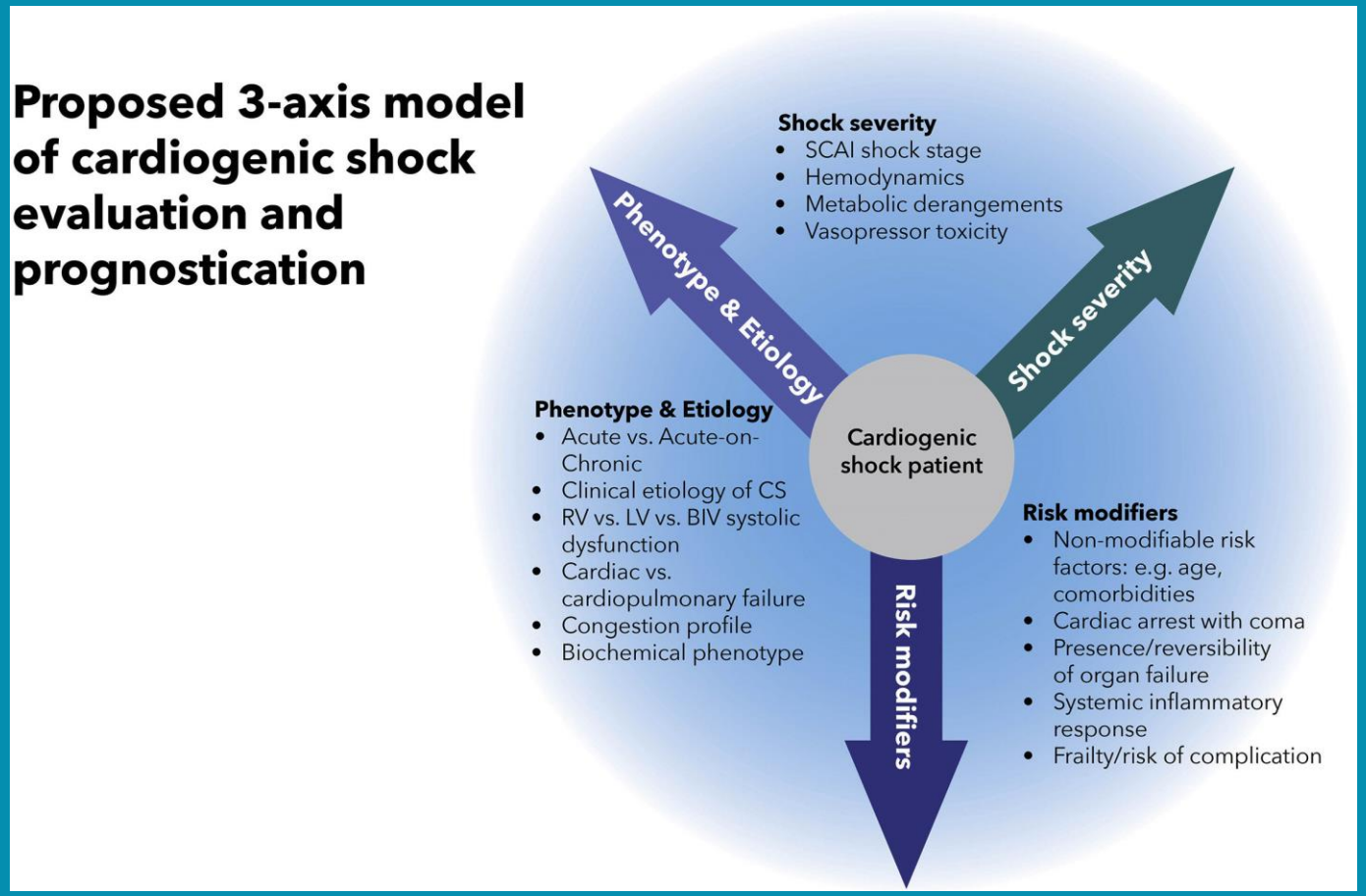
Journal of the Society for Cardiovascular Angiography & Interventions 2022 1DOI: (10.1016/j.jscai.2021.100008)

SCAI SHOCK stage in the context of acuity of presentation, etiology, phenotype, and other risk modifiers—the 3-axis model



Journal of the Society for Cardiovascular Angiography & Interventions 2022

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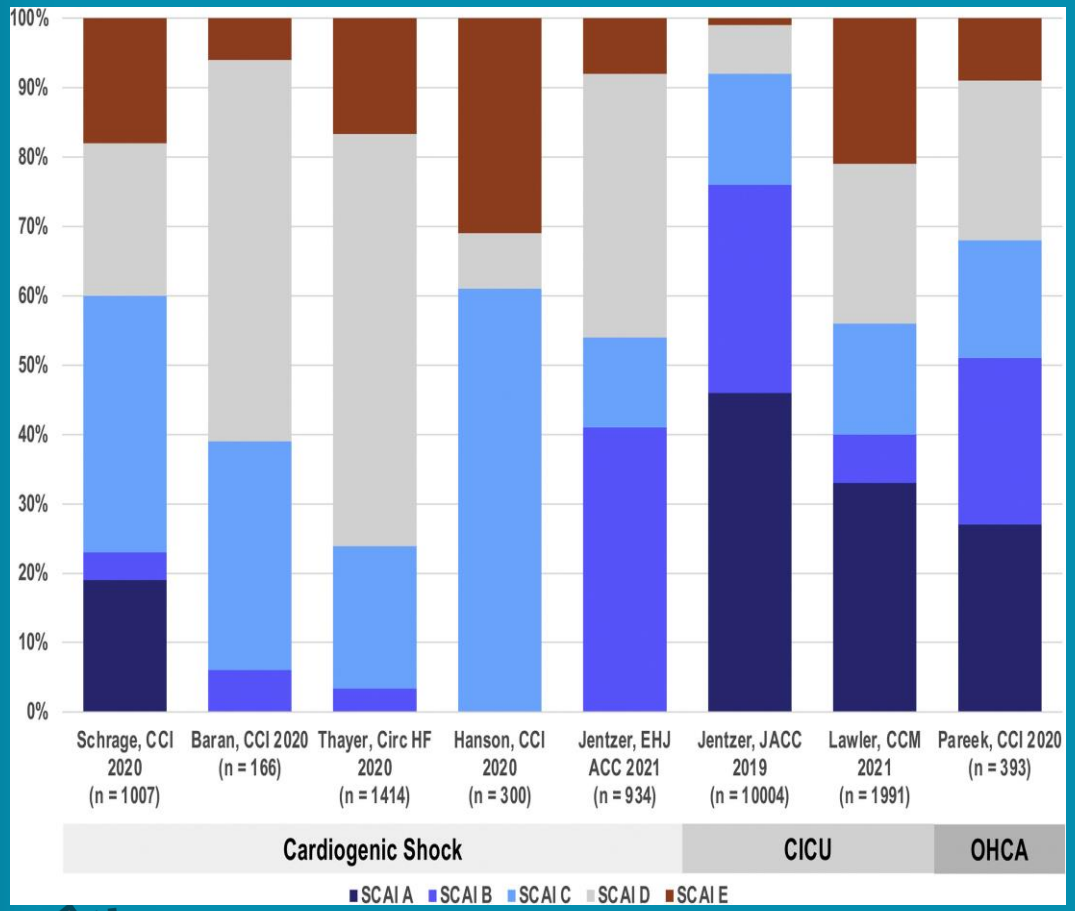
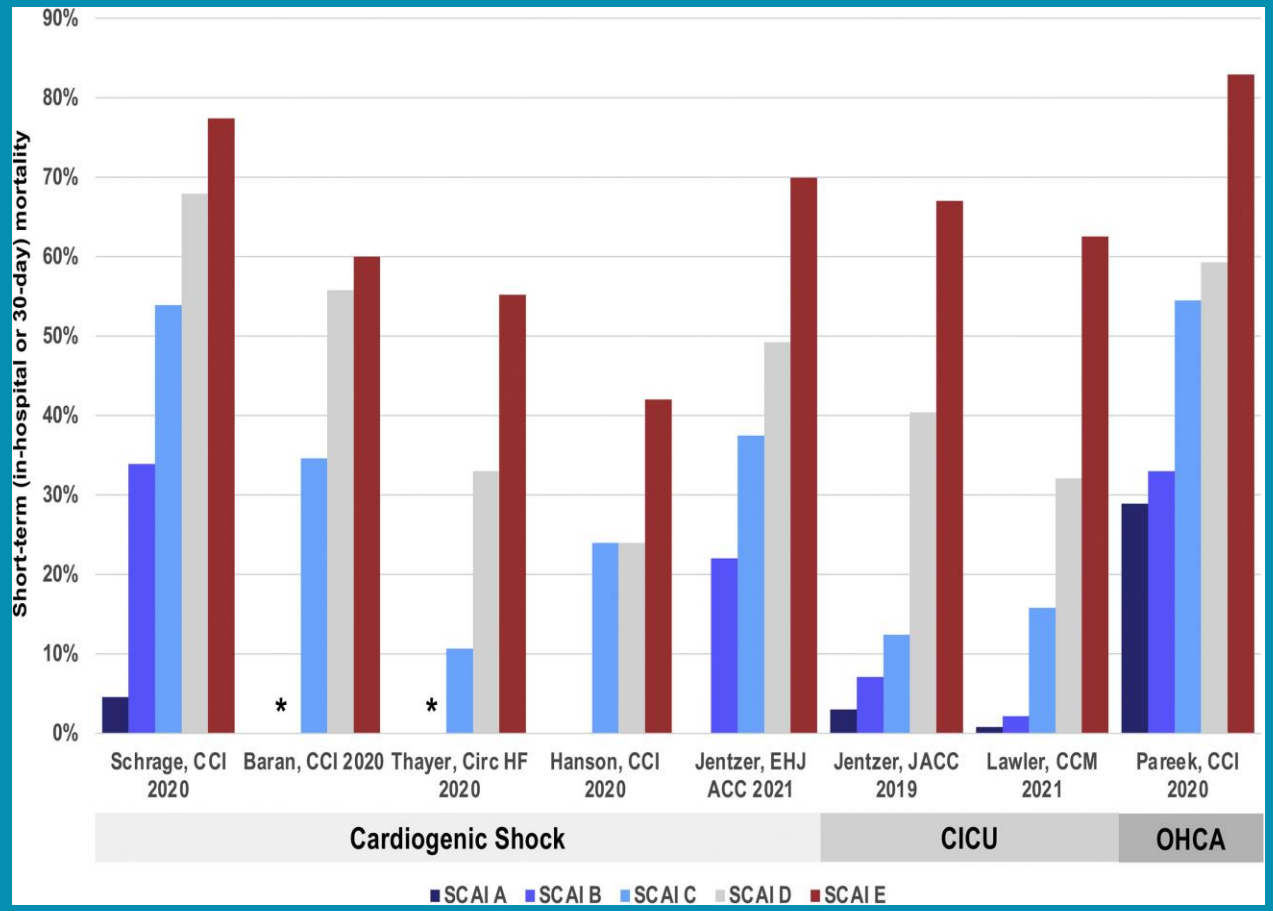
Summary of published SCAI SHOCK validation studies





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Journal of the Society for Cardiovascular Angiography & Interventions 2022 1DOI: (10.1016/j.jscai.2021.100008)

Etiology

TABLE 1 Common Etiologies of Cardiogenic Shock

Left ventricular failure

- Acute myocardial infarction
- Hypertrophic obstructive cardiomyopathy
- Myocarditis
- Myocardial contusion
- Peripartum cardiomyopathy
- Post-cardiotomy
- Progressive cardiomyopathy
- Septic cardiomyopathy
- Stress cardiomyopathy (takotsubo)
- Ventricular outflow obstruction

Right ventricular failure

- Acute myocardial infarction
- Myocarditis
- Post-cardiotomy
- Progressive cardiomyopathy
- Pulmonary embolism
- Septic cardiomyopathy
- Worsening pulmonary hypertension

Arrhythmia

- Atrial fibrillation or flutter
- Ventricular tachycardia or fibrillation
- Bradycardia or heart block

Pericardial disease

- Tamponade
- Progressive pericardial constriction

Chemotherapeutic, toxic, metabolic

- Calcium-channel antagonists
- Adrenergic receptor antagonists
- Thyroid disorders

Valvular or mechanical dysfunction

- Aortic regurgitation—acute bacterial endocarditis
- Mechanical valve dysfunction or thrombosis
- Mitral regurgitation—myocardial ischemia or infarction
- Progressive mitral stenosis
- Progressive aortic stenosis
- Ventricular septal defect or free wall rupture

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Tehrani B, Truesdell A, Psotka M, et al. A Standardized and Comprehensive Approach to the Management of Cardiogenic Shock. *J Am Coll Cardiol HF.* 2020 Nov, 8 (11) 879–891.

Management-Stabilization and Resuscitation Strategy

- IVF--- CAREFULLY
- OXYGENATION
- VENTILATION
- VASOPRESSORS
- CRRT
- HEMODYNAMIC MONITORING

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Hemodynamic monitoring

Type	CO	HR	CVP	PCWP	SVR	02 Sat
Cardiogenic	↓	↑	↔ ↑	↑	↑	↓
Hypovolemic	↓	↑	↔ ↑	↓	↑	↓
Septic	↑	↑	↔ ↑	↓	↓	↑
Neurogenic	↓	↓	↔ ↑	↓	↓	↓
Anaphylactic	↓	↑	↔ ↑	↓	↓	↓

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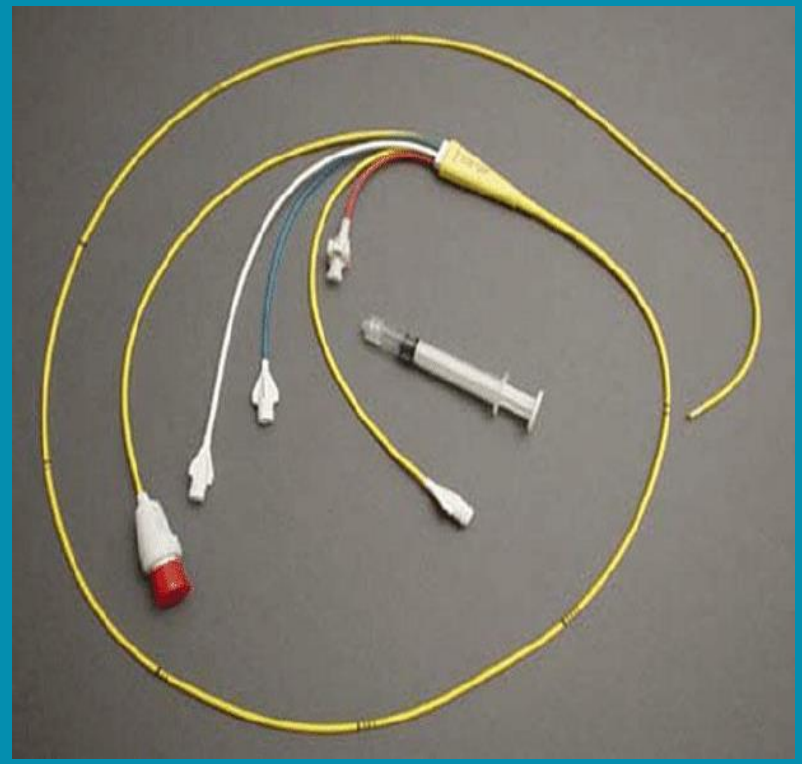
PA Catheter

- RAP, RVP, PAP, PCWP
- PVR
  - MEAN PAP-PCWP/CO– NORMAL < 1.5 WU
- SVR
  - MAP-RAP/CO X80—NORMAL 800-1200 DYNE.S.CM-5
- PAPI
  - PAS-PAD/CVP-NORMAL >2.0
  - < 1.0 IN AMI= 100% SENSITIVITY AND 98% SPECIFICITY IN PREDICTING IN HOSPITAL MORTALITY OR NEED FOR RVAD
- RAP/PCWP RATIO
  - NORMAL < 0.6; RATIO > 0.8 PREDICTS RVF IN AMI
- CARDIAC OUTPUT/INDEX
- CPO
  - MAPXCO/451



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ELSEVIER

Journal of the American College of Cardiology

Volume 44, Issue 2, 21 July 2004, Pages 340-348

Clinical research: cardiogenic shock

Cardiac power is the strongest hemodynamic correlate of mortality in cardiogenic shock: A report from the SHOCK trial registry ☆

Rupert Fincke MD *, Judith S. Hochman MD, FACC †, April M. Lowe MS ‡, Venu Menon MD, FACC §, James N. Slater MD, FACC †, John G. Webb MD, FACC ‖, Thierry H. LeJemtel MD, FACC ¶, Gad Cotter MD, FACC #, SHOCK Investigators

hospital mortality with a CPO ≤0.53 W was 58% (positive predictive value), whereas the probability of survival given a CPO >0.53 W was 71% (negative predictive value).

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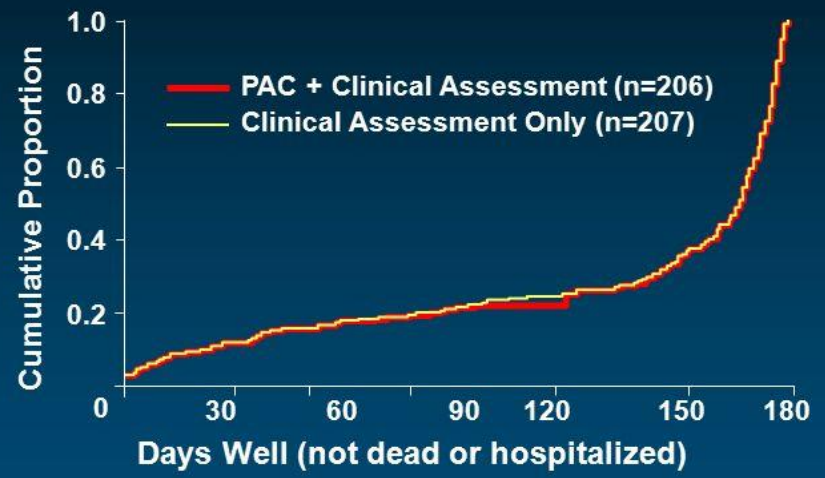
Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness

The ESCAPE Trial

Use of Pulmonary Artery Catheterization: No Effect on Mortality and Hospitalization (ESCAPE)



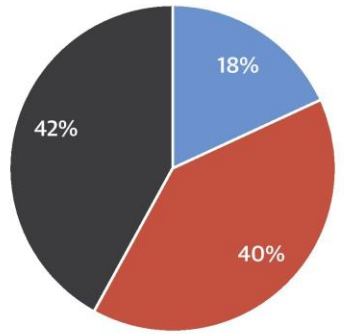
Reproduced with permission from The ESCAPE Investigators and ESCAPE Study Coordinators. *JAMA*. 2005;294:1625-1633.

Complete Hemodynamic Profiling With Pulmonary Artery Catheters in Cardiogenic Shock Is Associated With Lower In-Hospital Mortality

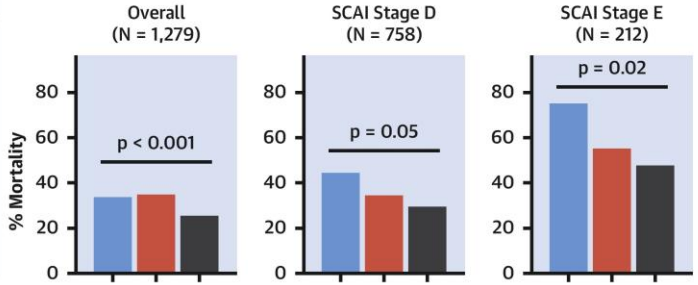
CENTRAL ILLUSTRATION: Frequency of Mortality Among PAC Use Overall and by SCAI Stage

PAC Utilization	Study Definition
None	Presence of **NONE** of the following invasive hemodynamics: • Pulmonary Artery Systolic Pressure • Pulmonary Artery Diastolic Pressure • Pulmonary Capillary Wedge Pressure • Pulmonary Artery Saturation • Right Atrial Pressure
Incomplete Assessment	Presence of **1-4** of the following invasive hemodynamics: • Pulmonary Artery Systolic Pressure • Pulmonary Artery Diastolic Pressure • Pulmonary Capillary Wedge Pressure • Pulmonary Artery Saturation
Complete Assessment	Presence of **ALL** of the following invasive hemodynamics: • Pulmonary Artery Systolic Pressure • Pulmonary Artery Diastolic Pressure • Pulmonary Capillary Wedge Pressure • Pulmonary Artery Saturation **AND** presence of Right Atrial Pressure

PAC Utilization Among Study Cohort



Association with Mortality Among Advanced Stage Patients



Garan, A.R. et al. J Am Coll Cardiol HF. 2020;8(11):903-13.

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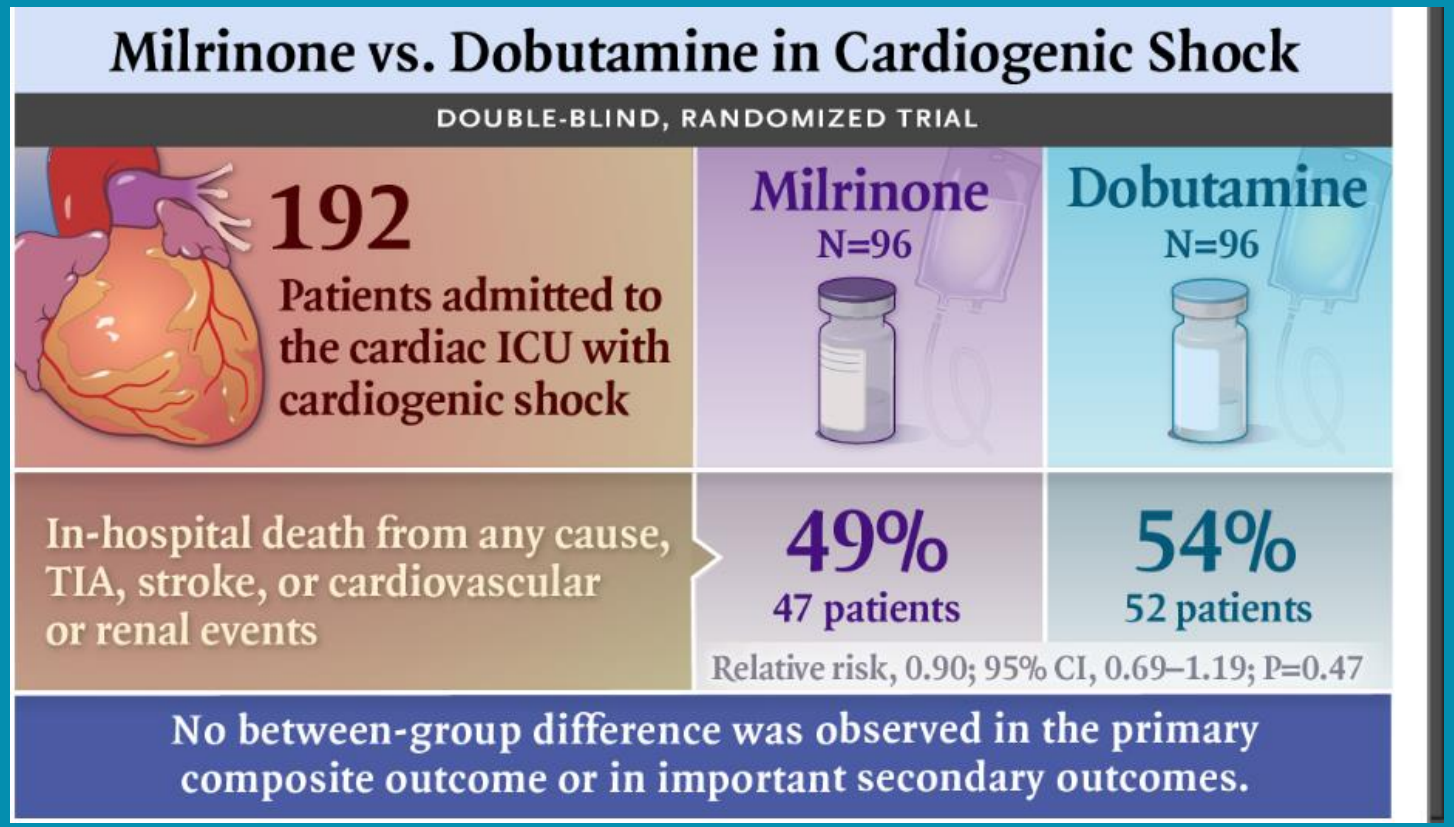
Medical Treatment

- TARGET IN GENERAL IS MAP >65 MM HG
- GOAL OF THERAPY IS TO INCREASE CONTRACTILITY AND DECREASE AFTERLOAD
- DEFINITIVE EVIDENCE SUPPORTING THE USE OF ONE SPECIFIC AGENT LACKING
- AIM FOR LOW TO MODERATE DOSES OF COMBINATION OF MEDICATION TO AVOID EXCESSIVE ALPHA-ADRENERGIC STIMULATION TO DECREASE END ORGAN ISCHEMIA
- FIRST LINE- USUALLY NOREPINEPHRINE

Milrinone as Compared with Dobutamine in the Treatment of Cardiogenic Shock

Rebecca Mathew, M.D., Pietro Di Santo, M.D., Richard G. Jung, Ph.D., Jeffrey A. Marbach, M.B., B.S., Jordan Hutson, M.D., Trevor Simard, M.D., F. Daniel Ramirez, M.D., David T. Harnett, M.D., Anas Merdad, M.B., B.S., Aws Almufleh, M.B., B.S., Willy Weng, M.D., Omar Abdel-Razek, M.D., et al.



N Engl J Med 2021; 385:516-525DOI: 10.1056/NEJMoa2026845

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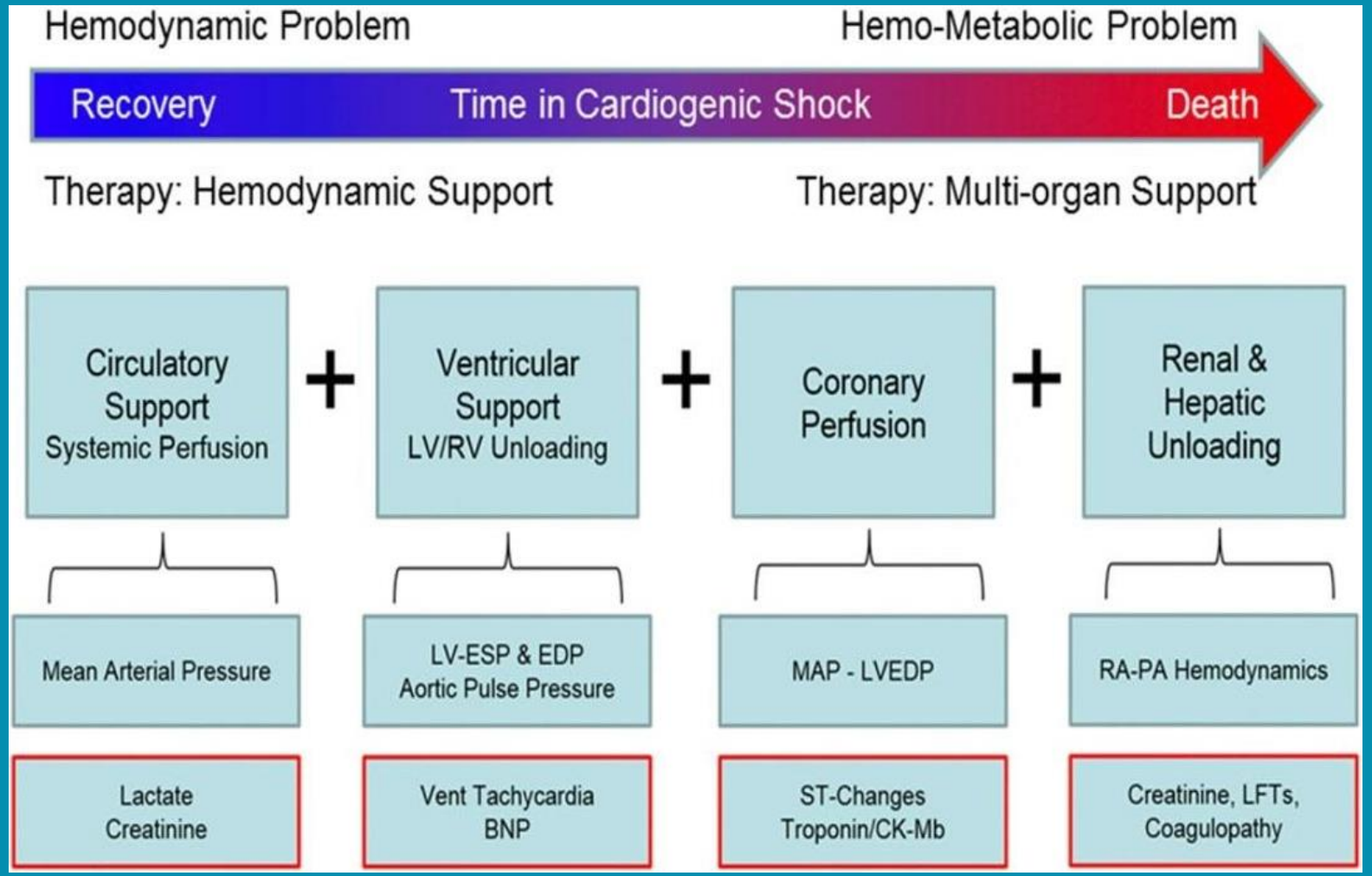
REVASCULARIZATION

Trial	n	Study Type	Objective	Primary Outcome Measures	Results
Medical Treatment and Interventional Trials in CS					
SHOCK 1999[6]	302	RCT MC	Emergency revascularization versus initial medical stabilization in AMICS	30-day all-cause mortality Secondary endpoint: 6-month survival	No difference in 30-day mortality Significant survival benefit after 6 months
SHOCK White et al. 2005[80]	302	RCT MC	Subgroup analysis of SHOCK trial: comparison of PCI versus CABG for early revascularization	30-day and 1-year survival	No difference in 30-day or 1-year survival
TRIUMPH 2007[81]	398	RCT MC	Effect of tilarginine acetate in AMICS	30-day all-cause mortality	No mortality reduction Study terminated after 398 patients
SHOCK-2 2007[82]	79	RCT MC	l-n-monomethyl-arginine (l-NMMA), a non-selective nitric oxide synthase inhibitor, versus placebo in AMICS	Absolute change in mean arterial pressure (MAP) at 2 h	l-NMMA resulted in modest increases in MAP at 15 min compared with placebo, but there were no differences at 2 h
Fuhrmann et al. 2008[83]	32	RCT SC	Levosimendan versus enoximone on top of PCI, IABP and inotropes in refractory CS due to acute MI	30-day all-cause mortality	Improved survival in levosimendan group
SOAP-2 2010[77]	1,679	RCT MC	Dopamine versus norepinephrine in the treatment of shock	28-day all-cause mortality	No mortality difference Dopamine: greater number of adverse events Subgroup of CS: Increased mortality when treated with dopamine
PRAGUE-7[84]	80	RCT MC	Abciximab pre/post PCI versus control	Combined: death, reinfarction, stroke, or new renal failure at 30 days	No benefit of abciximab
CULPRIT SHOCK 2017[36]	706	RCT MC	PCI of culprit lesion alone versus immediate multivessel PCI	Composite of death or severe renal failure leading to renal replacement therapy within 30 days	PCI of culprit lesion is superior to multivessel PCI regarding the composite endpoint
OptimaCC 2018[78]	57	RCT MC	Epinephrine versus norepinephrine for AMICS	Cardiac index evolution Primary safety outcome was the occurrence of refractory CS	Epinephrine compared with norepinephrine was associated with similar effects on arterial pressure and cardiac index and a higher incidence of refractory shock
SHOCK COOL[75]	40	RCT SC	Mild hypothermia (33°C) in AMICS versus control	Cardiac power index at 24 hours Secondary endpoint: 30-day mortality	No difference in cardiac power index No difference in 30-day mortality

Cardiogenic Shock Management and Research: Past, Present, and Future Outlook, *US Cardiology Review 2022;16:e03.*

LV Unloading



Esposito ML, Kapur NK. Acute mechanical circulatory support for cardiogenic shock: the "door to support" time. F1000Res. 2017 May 22;6:737

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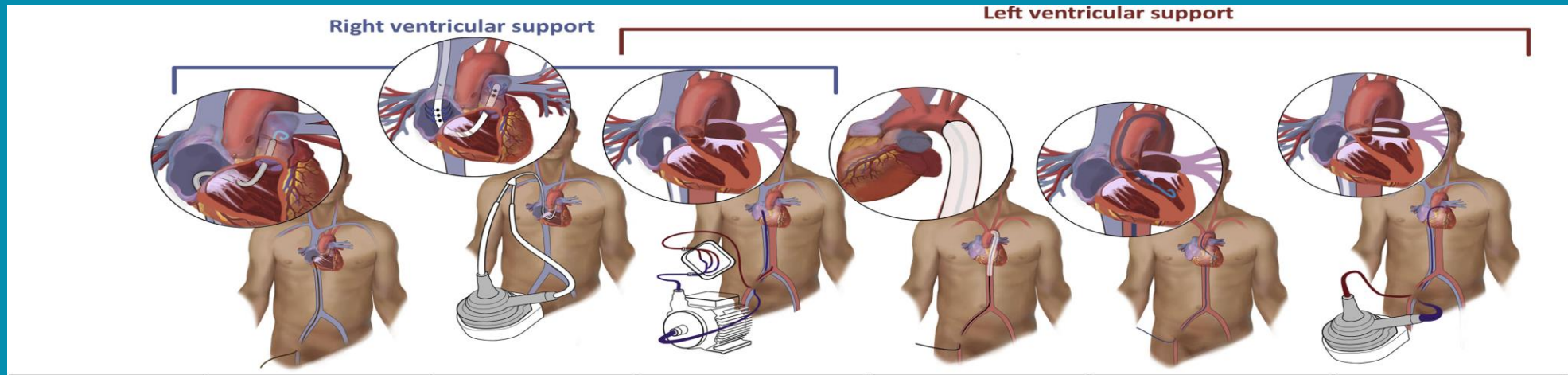

	Impella RP	TandemHeart RA-PA	VA-ECMO	IABP	Impella (2.5, CP, 5.0, 5.5)	TandemHeart LA-FA
Flow	max 4.0 l/min	max 4.0 l/min	max 7.0 l/min	0.5 l/min	2.5 - 5.5 l/min	max 4.0 l/min
Pump Speed	33000 rpm	max 7500 rpm	max 5000 rpm	NA	max 51,000 rpm	max 7500 rpm
Mechanism	Axial flow continuous pump (RA-to-PA)	Centrifugal flow continuous pump (RA-to-PA)	Centrifugal flow continuous pump (RA-to-AO)	Balloon inflation-deflation (AO)	Axial flow continuous pump (LV-to-AO)	Centrifugal flow continuous pump (LA-to-AO)
Cannula Size	22 F venous	29 F venous	14-19 F arterial 17-21 F venous	7-8 F arterial	13-21 F arterial	12-19 F arterial 21 F venous
Insertion/Placement	Femoral vein	Internal jugular vein	Femoral vein Femoral artery	Femoral artery Axillary artery	Femoral artery Axillary artery	Femoral artery Femoral vein
LV Unloading	-	-	-	+	+ to +++	++
RV Unloading	+	+	++	-	-	-
Cardiac Power	-	-	↑↑	↑	↑↑	↑↑
Afterload	-	-	↑↑	↓	↓↓	↑
Coronary Perfusion	-	-	-	↑	↑	-
Considerations	• *RECOVER RIGHT: 73% survival-to-30 days in RVF post LVAD, AMI or cardiotomy* • *May 2019 - FDA post-approval study: 33% survival-to-30 days*	• *IJ access may facilitate early ambulation*	• *Bi-V + oxygenation support for CS following:* - *AMI, ADHF or cardiac arrest* - *Cardiotomy* - *Myocarditis* - *Allograft rejection*	• *Requires stable cardiac rhythm and native heart function* • *May consider in select cases of post-AMI mechanical complications*	• *June 2008 – FDA 510(k) approval for HR-PCI* • *April 2016: Expanded Indication for CS* • *Contraindicated with mechanical aortic valve, LV thrombus*	• *Requires transseptal access* • *Oxygenator may be added to the circuit*

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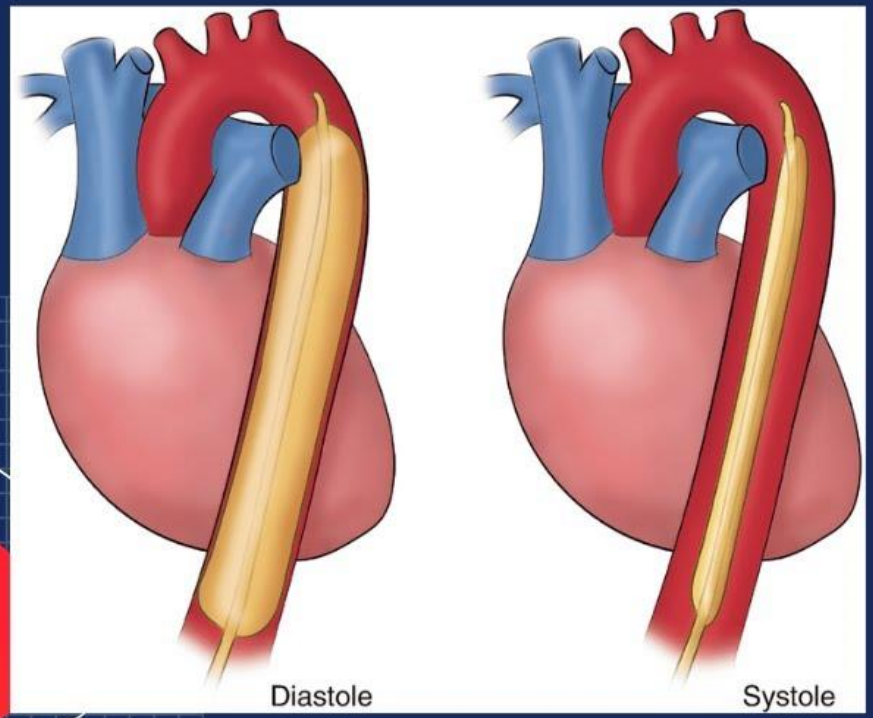
Tehrani B, Truesdell A, Psotka M, et al. A Standardized and Comprehensive Approach to the Management of Cardiogenic Shock. *J Am Coll Cardiol HF.* 2020 Nov, 8 (11) 879–891.

INTRA-AORTIC BALLOON PUMP



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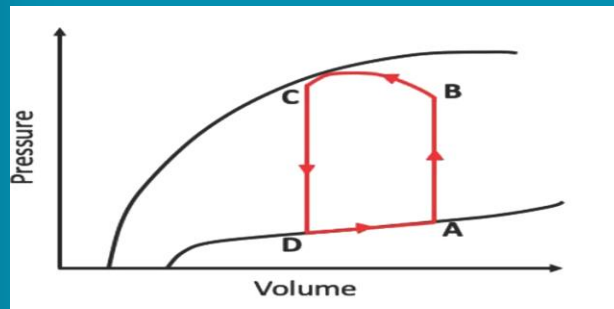
Impella

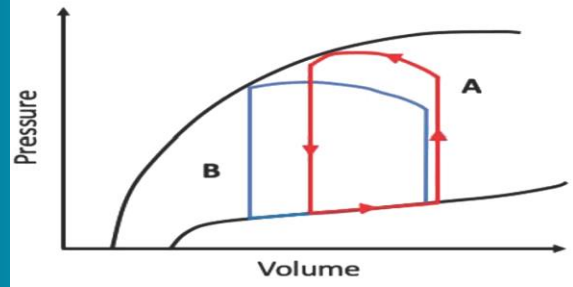


Work = Pressure x Volume

Ventricular "Work" = Area of PV Loop; proportional to O2 demand

Unloading Work = Reducing Area of PV Loop

A = End diastole (mitral valve closure)
B = Aortic valve opening
C = End systole (aortic valve closure)
D = Mitral valve opening

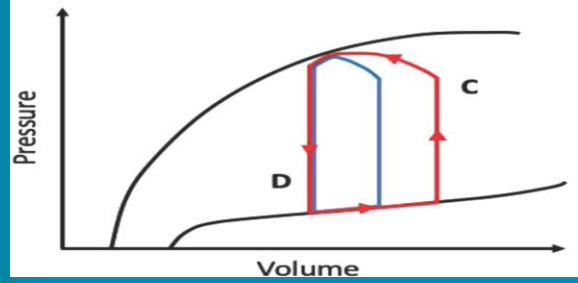


IABP

- Reduces systolic aortic pressure
- Increases stroke volume

Effect on Cardiac Work = Stroke Volume increase offsets pressure reduction

A = Baseline PV loop
B = After IABP

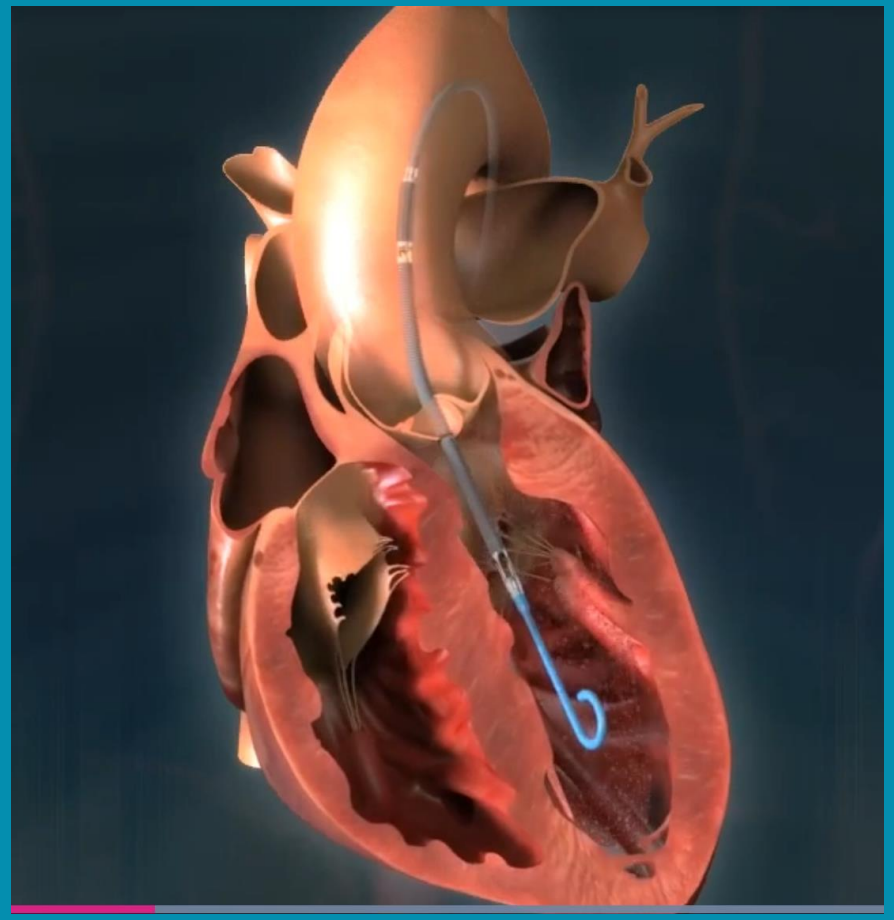


Impella

- Unloads left ventricle
- Reduces diastolic volume

Effect on Cardiac Work = Volume reduction reduces PV loop area and cardiac work

C = Baseline PV loop
D = After Impella



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Mechanical Circulatory Support Trials in CS					
Thiele et al. 2005[85]	41	RCT SC	IABP versus TandemHeart (TH) in AMICS	Cardiac power index	TH: improved cardiac power index TH: more bleeding and limb ischemia – no difference in 30-day mortality
Burkhoff et al. 2006[86]	33	RCT MC	IABP versus TandemHeart in AMICS	30-day all-cause mortality	No difference in 30-day mortality
ISAR-SHOCK 2008[44]	26	RCT MC	IABP versus Impella 2.5	Cardiac index 1 h after device implantation	Impella: improved cardiac index No difference in 30-day mortality
IABP-SHOCK II 2012[87]	600	RCT MC	IABP versus standard care	30-day all-cause mortality	No mortality reduction due to IABP
IMPRESS 2016[47]	48	RCT MC	IABP versus Impella CP	30-day all-cause mortality	No difference in 30-day mortality or after 6 months Impella: more major bleeding
Basir et al. 2019[30]	171	p-Coh	Hemodynamic monitoring and early MCS with Impella in AMICS	Survival to discharge	Standardized shock protocol and early MCS with Impella is associated with improved survival
Pozzi et al. 2020[62]	56	r-Coh SC	VA-ECMO in AMICS	Survival to discharge	Survival-to-discharge rate 41%
Lemor et al. 2020[55]	6,290	r-Coh MC PSM	Impella (n=5,730) versus V-A ECMO (n=569) in AMICS	In-hospital mortality	Lower mortality with Impella than with VA-ECMO
Dhruva et al. 2020[53]	3536	r-Coh MC PSM	Impella versus IABP in AMICS	In-hospital mortality Major bleeding	Increased risk of in-hospital death and major bleeding with Impella compared with IABP
ARREST 2020[73]	30	RCT SC	Early ECMO-facilitated resuscitation versus standard ACLS treatment	Survival to discharge	ECMO group: significantly improved survival
Varshney et al. 2020[88]	55	Case series	Impella 5.5 in AMICS	Survival to explant Recovery of native heart function	Survival to explant: 83.6% Recovery of native heart function: 76.1%
ECLS-SHOCK 2020[64]	41	RCT SC	VA-ECMO versus standard care in AMICS	Left ventricular ejection fraction after 30 days Secondary: 1-year mortality	No decrease in 1-year mortality with V-A ECMO Study was not powered to assess mortality
Schrage et al. 2020 [72]	510	r-Coh MC PSM	LV unloading with Impella versus no unloading in patients treated with VA-ECMO for CS	30-day all-cause mortality	LV venting: lower all-cause mortality but more severe bleeding

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Cardiogenic Shock Management and Research: Past, Present, and Future Outlook, *US Cardiology Review 2022;16:e03.*

National Cardiogenic Shock Initiative

Study Design

- DESIGN: Prospective, non-randomized, single-arm, multi-center study
- OBJECTIVE: To assess the impact of early MCS, guided by invasive hemodynamics, on outcomes in AMICS, using the NCSI protocol.
- NCT03677180

July 2016 to November 2020
1103 patients screened at 80 centers

→ 697 patients excluded

406 patients enrolled

Inclusion Criteria Not Met*	
No PCI performed	231
No evidence of hypotension	36
No evidence of hypoperfusion (clinically or by invasive hemodynamics)	36
No evidence of AMI	24

Exclusion Criteria Met*	
IABP prior to Impella	195
Unwitnessed Arrest or ROSC >30 min	108
Other Shock	57
Active Bleeding	43
Mechanical Complication of AMI	29
Recent Major Surgery	21
LV Thrombus	10
Mechanical Aortic Valve	4

*more than one exclusion criteria can apply

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Mir Babar Basir DO, William O'Neill MD, on behalf of the NCSI Investigators

RAPID IDENTIFICATION OF CARDIOGENIC SHOCK

SBP <80 or < 90 with Vasopressors
End organ hypo perfusion

AMI
STEMI/NSTEMI

CATH LAB

LVEDP > 15

NO — Fluid

YES — Impella

Culprit lesions PCI

Right heart catheterization Hemodynamics

GDMT for AMI

CRITICAL CARE

Cardiogenic Shock Intervention team

ICU Management
CPO/PAPI
Lactate levels

CPO <0.6

- PAPI < 0.9 → Consider RV support
- PAPI >0.9 → Consider ↑LV support/ LVAD

Hypoxia → ECMO

CPO >0.6 and PAPI >0.9 → Wean Vasopressors and Impella support

Failure to wean → Destination LVAD/Transplant/ Palliative care

Shock Management Adjudication Committee

Non-ischemic
CHF

CRITICAL CARE TEAM

Perform Post-PCI Hemodynamic Calculations

1. Cardiac Power Output (CPO): $\frac{MAP \times CO}{451}$

2. Pulmonary Artery Pulsatility Index (PAPI): $\frac{sPAP - dPAP}{RA}$

RESOURCES

Aditya Mehra, MD, Director of Cardiogenic Shock
Tresy Thomas, ANP- Coordinator

Hackensack Meridian *Health*
Jersey Shore University Medical Center

Reference: National Cardiogenic Shock Initiative. Henry Ford Health System. Retrieved from: https://www.henryford.com/cardiogenicshock
Adapted from Hackensack University Medical Center Cardiogenic Shock Protocol

NCSI: Survival Outcomes

	All	C/D	E	p value
Procedural	99%	99%	98%	0.74
Discharge	71%	79%	54%	<0.01
30-days	68%	77%	49%	<0.01
1-Year	53%	62%	31%	<0.01

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Table 3: Ongoing Trials in Cardiogenic Shock

Name	n	Status	Study Type	Intervention
Medical Treatment Trials in CS				
COCCA (NCT03773822)	380	Recruiting	RCT MC	Combination of hydrocortisone + fludrocortisone versus placebo
DAPT-SHOCK-AMI (NCT03551964)	304	Recruiting	RCT MC	Comparison of intravenous cangrelor and oral ticagrelor in patients with acute MI complicated by initial cardiogenic shock and treated with primary angioplasty
ACCOST-HH (NCT03989531)	150	Recruiting	RCT MC	Adrecizumab versus placebo
Mechanical Circulatory Support Trials in CS				
EURO SHOCK (NCT03813134)	428	Recruiting	RCT MC	Early intervention with ECMO therapy or standard treatment with no ECMO
REVERSE (NCT03431467)	96	Recruiting	RCT MC	Patients randomized to the experimental arm will have an Impella-CP implanted in addition to VA-ECMO within a maximum of 10 hours of institution of VA-ECMO
ECMO-CS (NCT02301819)	120	Recruiting	RCT MC	Immediate VA-ECMO versus early conservative therapy according to standard practice
DanShock (NCT01633502)	360	Recruiting	RCT MC	Impella CP versus conventional circulatory support
ECLS-SHOCK (NCT03637205)	420	Recruiting	RCT MC	PCI (or CABG) plus medical treatment + extracorporeal life support in CS versus PCI (or CABG) plus medical treatment
UNLOAD-AMI (NCT04562272)	80	Recruiting	RCT SC	Mechanical unloading by Impella-CP for 36-48 hours, as add-on to the standard treatment versus standard treatment
SMART-RESCUE II (NCT04143893)	1,000	Recruiting	RCT SC	MCS + medical treatment versus medical treatment alone

CABG = coronary artery bypass graft; Coh = prospective cohort analysis; ECMO = extracorporeal membrane oxygenation; MC = multicenter; nyR = not yet recruiting; PCI = percutaneous coronary intervention; R = recruiting; RCT = randomized controlled trial; SBP = systolic blood pressure; SC = single center; VA-ECMO veno-arterial extracorporeal membrane oxygenation.

Conclusions

1. CS remains the most common cause of mortality in patients with AMI.
2. There was a stepwise increase in risk of hospital mortality with increments of SCAI shock stages A–E.
3. Classification, stabilization, and diagnostic evaluation of AMICS are prerequisites to tailored invasive therapy
4. Patients presenting in shock (stages C–E) may first require acute stabilization with attention to blood pressure, end organ perfusion status, oxygenation, and acid-base status.

1. AHA recommended the development of CS centers using standardized protocols for diagnosis and management of CS,
2. An individualized approach to care and participation in clinical research protocols to test the utility of MCS in AMICS is recommended.
3. Finally, optimization of care of AMI complicated by CS requires a multidisciplinary team effort

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Invasive Management of Acute Myocardial Infarction Complicated by Cardiogenic Shock: A Scientific Statement From the American Heart Association. Circulation 2021;Mar 4:

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